Mail

Aspartame and breast and other cancers

To the Editors,
I discovered an extraordinary correlation between asparatame (marketed as Nutrasweet and in its generic form) and increasing breast and prostate cancer incidence.

My observation occurred while I was researching the metabolism of aspartame. Aspartame consists of phenylalanine, aspartic acid and methanol (wood alcohol). Upon metabolism, Nutrasweet breaks down into aspartic acid, a neuroexcitatory agent,[1] phenylalanine, an amino acid, and methanol. The methanol is converted to formaldehyde,[2] which then accumulates within the cells.[3]

Formaldehyde has been considered an inducer of cancer[4] and acts to alter DNA.[5,6] Thus, it seemed reasonable to superimpose a graph of breast cancer incidence against that of the rising use of Nutrasweet (aspartame).

Nutrasweet received limited approval for use as a sweetener in 1974, then further expansion in 1981 and unlimited approval in 1983. These points are marked on the curve of breast cancer increases obtained from the governmental surveillance statistics (see Figure 1).

A similar relation was found with prostate cancer. Interestingly, breast and prostate cancer rates are five to six times higher in Europe

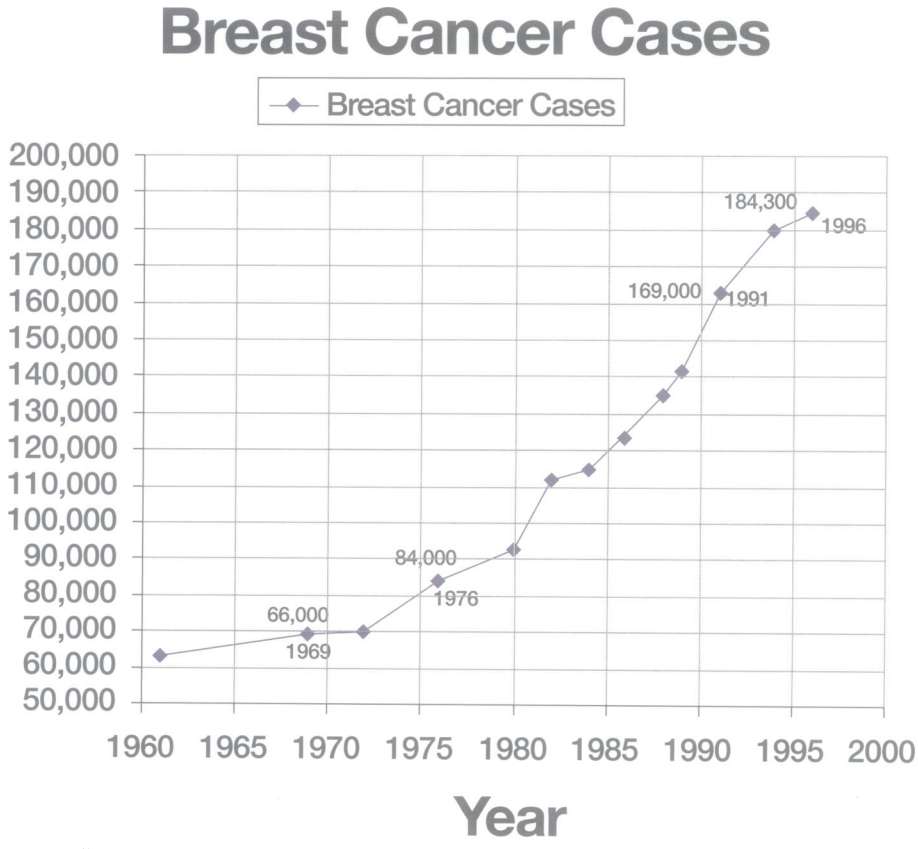

Figure 1 Illustration of breast cancer incidence based on government surveillance statistics

and North America than in Asia and Africa, the latter correlating with lower use of Nutrasweet.[7,8] Also, review of the original Nutrasweet animal laboratory research findings released under the Freedom of Information Act revealed mammary tumors were observed in many of the animals.

The correlation associated with a likely mechanism of cancer induction in susceptible people points to a need for immediate scrutiny of aspartame as an environmental cause of many thousands of cancer cases.

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Response

The letter by Dr Schwartz has not taken into account some important information. It is true that aspartame consists of phenylalanine, aspartic acid, and methanol, but even among high consumers of aspartame, the relative contribution of these compounds is trivial, less than 10% of the average human intake of these compounds from regular diet[1,2]. Moreover, aspartame was not commercially available until 1981, so that the compound could not have been in the market in 1974.

With respect to the graph in Dr Schwartz's letter, it is well known that factors unrelated to each other, but having similar time trends, be these breast cancer incidence, aspartame intake, number of cars of a particular maker, or commercial air traffic, do demonstrate what is described here as extraordinary correlation. In epidemiology, we use the term ecological fallacy. The reported increasing incidence of breast cancer and prostate cancer in the United States may be due partly to the increased intensity of screening by mammography and prostate specific antigen (PSA), respectively, although early life influences may have contributed to the secular trends of breast cancer.[3] In any case, those who want to hazard etiological statements on the basis of these ecological data should also take into account several points, including the fact that incidence is a rate and should take into account the population at risk, that there is a latency involved in most etiological factors, including those with growth enhancing potential, and that the few analytic studies of artificial sweeteners in relation to breast cancer have yielded null results.[4,5] Perhaps even more important in this instance, is the fact that breast cancer incidence in the United States has been declining during the last few years.[3,6,7] It would be just as inaccurate to hypothesize that long-term use of aspartame reduces the risk of this cancer.

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Dr Trichopoulos is responding on behalf of Monsanto. Dr Trichopoulos' department receives research funding from Monsanto.

References

1. Stegink LD. Aspartame metabolism in humans: acute dosing studies in aspartame: Physiology and biochemistry. Stegink LD, Filer LJ Jr, eds. New York: Marcel Dekker, 1984;504.
2. Food and Drug Administration. Aspartame: Commissioner's final decision. Fed Regist. 1981;46:38285-38308.
3. Adami H-O, Signorello LB, Trichopoulos D. Towards an understanding of breast cancer etiology. Semin Cancer Biol. 1998;8:255-262.
4. Ewertz M. Breast cancer in Denmark. Incidence, risk factors, and characteristics of survival. Acta Oncol. 1993;32:595-615.
5. Ewertz M, Gill C. Dietary factors and breast-cancer risk in Denmark. Int J Cancer. 1990;46:779-784.
6. Chu KC, Tarone RE, Kessler LG, et al. Recent trends in U.S. breast cancer incidence, survival, and mortality rates. J Natl Cancer Inst. 1996 Nov 6;88:1571-1579.
7. Smart CR, Byrne C, Smith RA, et al. Twenty-year follow-up of the breast cancers diagnosed during the Breast Cancer Detection Demonstration Project. CA Cancer J Clin. 1997;47:134-149.

Modern obstetrics and female life span

To the Editors,

Advances in pre- and perinatal care have significantly decreased the mortality of females such that now, in contrast to former times, the life span of women in the United States exceeds that of men (78.8 versus 72.4 years). To ascertain the effect of childbearing on females of a far lower creature who do not have the benefits of modern medical advances, my colleagues and I chose to study the fruit fly.

We selected Drosophila melanogaster of the type yellow forked attached and white apterous (Carolina Biological Supply Co, Burlington, NC*) because of ease of differentiation between the sexes and ease of handling. A virgin, newly emerged (less than 12 hr old) female and similar male fly were added to each of 100 4″ × 1″ vials containing 4 g of Instant Drosophila Mix* containing 10 ml of water and a few grains of yeast. The vials were kept in an incubator at 22° C, and the paired flies were allowed to mate and lay eggs for 15 days. The two parents were then removed.

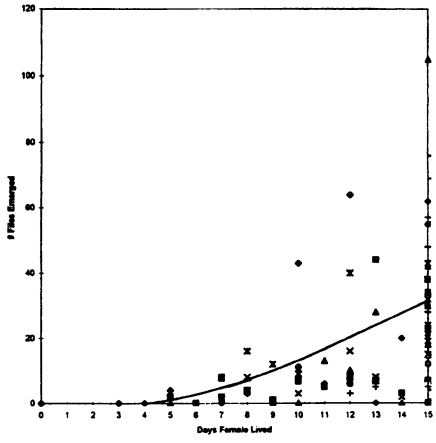

Figure 1 Number of flies emerged versus days female lived

During the 15-day interval, approximately twice as many females as male flies died (55 females, 33 males, p=0.0016). Indeed, we found that the mean life span of these flies was significantly shorter for the female than for the male during this interval (12.06 ± 0.49 versus 13.90 ± 0.29 days, p= 0.0019). After clearing, we counted (over an additional 2 weeks) the number of adult progeny flies that subsequently emerged in these vials. The data (Figure 1) show that females that live less than 15 days yield far less progeny than the heartier ones that were still alive on clearing day. These results indicate that child bearing does, indeed, take a toll on these primitive females and credit the obstetric advances that have alleviated this plight in human females.

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